A small prospective study by Vessey, et al (1986) of white married women ages 25-39 determined an overall incidence of ulcerative colitis of 0.15 per 1000 person/years and to 0.09 for Crohn's disease. Respective incidence rates in smokers were 0.11 (0.16 for 1-14 per day, 0.04 for >14 per day) and 0.17; in non-smokers they were 0.17 and 0.05. Incidence rates in ex-smokers were the same as in non-smokers for both diseases. Most importantly, the information on smoking habits was collected well prior to the onset of symptoms, making it highly unlikely that the disease influenced the women's smoking habits.

Certainly, the apparently increased risk of ulcerative colitis in ex-smokers and of Crohn's disease in current smokers is puzzling. One explanation for the increased colitis risk in ex-smokers contrasted to the decreased risk in current smokers is that the act of stopping smoking predisposes individuals to ulcerative colitis (Kennedy 1984, Amery 1984). Logan and Langman (1984) explored this hypothesis in their and others' data, finding no consistent supportive evidence. The results from Tobin, et al (1987) and Boyco, et al (1987) suggest that the apparently increased risk in ex-smokers might be artifactual, while a more recent study of colonic mucous production in vitro

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indicates that cessation of smoking disrupts the capacity of the normal colon for production of mucous (Cope 1988). The excess risk of Crohn's disease, however, appears real. Holdstock, et al (1984) suggest that smoking influences pathological appearances, perhaps by affecting the immune system, resulting in features of Crohn's disease rather than colitis.

Tobin, et al (1987) hypothesize that smoking might influence which disease develops in persons with genetic predisposition to inflammatory bowel disease. An explanatory biological mechanism for these suggestions remains elusive, although a mechanism for the observed colitis/smoking inverse relationship, i.e. a protective effect due to colonic mucus, seems quite plausible. In tissue culture, biopsied specimens from colitis patients had significantly less incorporation of tritiated glucosamine into colonic mucus. Non-smoking colitis patients showed reduced mucus production compared to non-smoking controls, while colitis patients who smoked had similar mucus production to that of all controls.

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Of course, some cause of ulcerative colitis might explain the observed association. Struck by the similarity of the inverse relationship in both Parkinson's disease and ulcerative colitis, Bihari and Lees studied twenty-five patients with both of these diseases (Bihari 1987). 20 never smoked, 3 were ex-smokers for a long time, 1 smoked a pipe occasionally, and 1 smoked 10 cigarettes per day. The authors call for prospective epidemiologic studies to determine whether persons with these two diseases, rather than sharing protection by smoking, instead share a "distinctive premorbid personality". This is unlikely, however, given the results of Monk, et al (1970) who, in a study of 193 ulcerative colitis patients, failed to determine psychological factors which were important in the development or continuation of the disease. Also, Helzer, et al (1982) conducted a personality assessment and tabulation of recent stressful events on fifty colitis patients and matched non-gastrointestinal controls, and found no greater frequency of diagnosable psychiatric disorder in the former. Personality profiles also were similar. Nor were significant differences found in comparisons between colitis and duodenal ulcer patients with regard to obsessive personality traits, although the colitis group worried more about their disease. No sociocultural

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factors were measured, although these are known to influence obsessive scores, and to be somewhat related to the disease, i.e. patients having higher educational and economic level than general population controls (Bellini 1976).

The scientific literature linking cigarette smoking to a significant reduction in the risk of ulcerative colitis is impressive for its congruence and the absence of other rationale explanations for this observation. While many may view this possible benefit of smoking as inconsequential in relation to its perceived overall effect on mortality, it is important to also consider how, in this case, smoking can improve the quality of life. Its affect appears to be strongest for current smokers, and may, in fact be limited to this group. Anecdotal reports imply reversibility of the protective action, with rapid effect. Missing is clear evidence of a plausible biological mechanism. Because of the speedy effect of smoking in ameliorating the symptoms of the disease, research to discover a mechanism for its action should be comparatively straightforward.

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PARKINSON'S DISEASE

The annual incidence of Parkinson's disease in the U.S. was estimated to be 16,000 - 36,000 new cases, with a (point) prevalence of 140,000 - 360,000 cases (Kessler 1973). Although the etiology of the disease is largely unknown, hence the designation idiopathic Parkinson's disease (IPD) for the most common variety of Parkinson's syndrome, results from most epidemiologic studies of IPD demonstrate an inverse relationship between IPD and cigarette smoking.

Prospective follow-up studies of British doctors (Doll 1976, 1980), U.S. veterans (Kahn 1966) and over one million people in 25 states (Hammond 1966), found lower mortality rates from Parkinson's disease among persons who ever smoked compared to those who never smoked. Relatively few subjects in these studies developed Parkinson's, but in each investigation, the relative risk of mortality from IPD was lower in smokers. Of course, these studies are somewhat limited because Parkinson's disease is not usually an immediate cause of death and its reporting on death certificates is

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unreliable. Thus, these prospective studies do not give the complete picture of the relationship between smoking and PD. Furthermore, no data on temporal sequence or potentially confounding factors were available.

Case-control studies provide larger numbers of IPD subjects, and therefore, more precise risk estimates, although the potential for bias is greater. Early community and hospital-based studies by Kessler (Kessler 1972a, 1972b, 1973) confirmed the findings from the prospective studies. More recently, Rajput, et al (1987) compared smoking histories of 64 female and 54 male cases with those of 128 female and 108 male controls. 45% of the controls reported having ever smoked, compared to 37% of cases; a result suggestive of an negative relationship, but not statistically significant (OR = 0.7, 95% CI = 0.4-1.2). Former smokers were more frequent among cases than controls (24% vs 19%), but current smokers were twice as frequent among controls than cases (26% vs 13%). The relative risks for ever-smokers compared to never-smokers decreased with increasing age, ranging from 2.5 for the 45-59 age group to 0.3 for the 80+ group. Neither the relative risks nor the trend were statistically significant.

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The mean age at diagnosis was significantly older for the never-smoked group than for the ever-smoked group. Since smoking history was obtained at the time of diagnosis, and inability to recall previous smoking habits may be associated with the disease, recall bias might have been present.

Godwin-Austen, et al (1982) conducted a case-control study of 350 pairs of Parkinson's patients and matched controls. An odds ratio of 0.5 for current smoking vs non-smoking was found to be statistically significant. The risk was the same regardless of whether smoking status at the time of diagnosis or 20 years earlier was used. Relative risks for ex-smoking vs never smoking decreased from 0.8 for those who quit 10 years before diagnosis, to 0.6 for those who quit 1-10 years before diagnosis.

In a Finnish case-control study of 443 Parkinson's disease patients and age- and sex-matched controls, 73.6% of the PD patients had ever smoked compared to 67.3% of the controls, a statistically significant difference (Marttila 1980). Furthermore, of the 117 PD patients who had smoked, 76.1% had stopped, while in the control series,

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significantly fewer (53.8%) had quit. However, the report does not mention when, in relation to the onset of symptoms, these persons quit smoking.

Baumann, et al (1980) compared smoking histories of Parkinson's disease patients and matched controls selected by door-to-door canvassing of the cases' neighbors. Because of this selection process, cases and controls were equivalent in terms of socioeconomic status and marital status, in addition to age and sex, for which they were matched. Information on coffee and alcohol consumption was collected and in both groups the percentages who had at some time regularly consumed coffee or alcoholic beverages were equivalent. Cases tended to begin drinking both beverages at a significantly earlier age than their neighbors. Of the Parkinson's disease patients, 63% had never smoked cigarettes regularly, compared to 47% of the controls; a statistically significantly reduced odds ratio of 0.46. This significant difference was evident for smoking status 20 years before symptoms onset, 10 years before, and just before onset. One explanation for these results, i.e. selective mortality of smoking Parkinson's patients could not be supported by the information

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collected on deceased patients, nor by differential proportions of smokers among patients with different disease durations. Nicotine exposure was determined from information on brands smoked, and was not significantly different between cases and controls.

Golbe, et al (1986) failed to find an inverse dose-response relationship between several measures of disease severity and cigarette usage in a study of 32,000 of the United Parkinson Foundation members. The study was irreparably flawed because the underlying hypothesis, i.e. "that patients with PD who have smoked should have milder disease and/or a late onset of symptoms than lifelong non-smokers," does not rule out a protective role for cigarette smoking. Furthermore, the scientific literature contains numerous examples which demonstrate that the age-of-onset for a disease may not be dose-related, even though the risk of the disease is thus related. Thus, while it appears from this study that the rate of disease progression and the age at diagnosis are unrelated to smoking status among people with PD, the study presents no evidence that smokers are more or less likely than non-smokers to get the disease in the first place.

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One case-control study (Rajput 1984) failed to confirm the inverse relationship between smoking and PD. In this study, medical records of Olmsted County, Minn., residents during 1967-1979 were reviewed for evidence of Parkinson's disease. For each case, the next two medical facility attendees matching the case in age and sex were selected as controls. Information on smoking habits and other illnesses was abstracted from the medical records. Approximately 55% of the controls and 52% of the patients never smoked. Controls reported significantly more hypertension and significantly less psychoneuroses and psychosomatic illnesses. However, information on tobacco usage is not reliably obtained from medical records, and controls selected from medical populations tend to include more smokers. Furthermore, the comparison populations in this study clearly were different in a number of factors which could have confounded the results, as manifested in differential hypertension prevalence. These factors probably account for the disparate findings.

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In studies with small numbers of subjects, the consistency of results across studies is a good indicator of the validity of an hypothesized relationship. In the aforementioned studies the magnitude of the risks is relatively consistent; risks are lower for current, as opposed to former smokers; and alternative explanations for studies' observations are comparatively unlikely. Furthermore, reasonable biological mechanisms exist for explaining how smoking could prevent or ameliorate Parkinson's disease or its symptoms. Most favored among these is the facilitation of dopamine release and dopaminergic neural transmission through chronic nicotine exposure (Baron 1986). (Most idiopathic parkinsonism features depletion of dopamine in the basal ganglia.) The findings of Baumann, et al (1980) and Haack, et al (1981) of no relation between nicotine exposure and the severity of disease among PD cases argues against this, but Haack, et al based their inferences on observations of cases only, and mistakenly assumed that the severity or age-at-onset of a disease necessarily correlates with the magnitude of an exposure. In any event, nicotine exposure determination has been shown to be rather imprecise (Gori 1986). Alternatively, it has been hypothesized that smoking lowers monoamine oxidase B (MAOB)

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activity, thus reducing the conversion of non-toxic precursors into a specific neurotoxin for dopaminergic nigrostriatal neurons. This hypothesis has received considerable attention recently with the finding of a nicotine-like pyridine derivative (MPTP) contaminating some illicit drugs, which, after conversion into MPPT by MAOB, kills these neurons and produces lasting symptoms of Parkinson's disease in humans and lower primates. However, Yong and Perry (1986) measured MAOB in autopsied brain tissues of PD patients and in adult controls dying without PD, and found enzyme activities similar. They did, on the other hand, find that MAOB activity in platelets of heavy smokers was significantly lower than that in non-smokers. In an experimental study, they found that hydrazine, a substance found in tobacco smoke, protected dopaminergic nigrostriatal neurons in mice from damage by MPTP.

Other possible mechanisms include blocking the uptake of (MPP^+-like) toxins into dopamine neurons by pyridines contained in cigarette smoke (Javitch 1984, Snyder 1985), creation by carbon monoxide of a reducing environment that protects the substantial nigra from oxidative damage (Calne 1983), or enhancement of the

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metabolism and elimination of toxic compounds by induction of cytochrome P450 systems in the liver or elsewhere (Baron 1986). These mechanisms require continued cigarette smoking, and thus are consistent with the evidence from Rajput, et al (1987) and Godwin-Austen, et al (1982), showing a lower risk for current smokers versus former smokers.

Other independent evidence, such as lower incidence of Parkinson's in blacks, (who smoke more than whites) and the apparently increasing number of cases occurring in younger age groups, (where the proportion of smokers has been decreasing) (Teravainen 1986) lends credence to this inverse association. There are, however, alternate, though somewhat unlikely, explanations for these findings. It is possible that persons predisposed to Parkinson's disease do not begin smoking because of sensitivity to the negative effects of repeated nicotine on metabolism (Maker 1987). Thus, the reason why fewer smokers appear among Parkinson's patients is that they, to a greater extent than non-predisposed persons, are unable to begin the smoking habit. This predisposition hypothesis implies an early familial or environmental influence on the etiology

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of the disease, a genetic component such as that which has been raised ever since James Parkinson first described "Paralysis Agitans" in 1817 (Barbeau 1982), or that "premorbid non-smoking in parkinsonian patients is simply an epiphenomenon of a premorbid PD personality type" formed before adolescence but not hereditary (Golbe 1986, Duvoisin 1981).

Several studies among twins clearly indicate no genetic component. Duvoisin, et al (1981) found zero concordance for Parkinson's disease among 12 monozygotic twin pair volunteers solicited through physicians and PD organizations in New York. In this study, 2 twins with Parkinson's disease (probands) and 5 twins without PD (co-twins) were current smokers. 4 probands and 1 co-twin were ex-smokers. Co-twins smoked for a longer period of time and more heavily, though none of the smoking differences approached statistical significance. Ward, et al (1983) studied 65 pairs of twins and a set of quadruplets, similarly solicited, and found a PD concordance rate of between 2% and 5%, depending on interpretation of inclusion criteria. They failed to find any postnatal etiologic factor, although they did confirm the differential smoking habits found by

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Duvoisin, et al. Further analysis of these twin pairs which concentrated on those discordant for PD found no statistically significant difference in possible environmental determinants. The only significant result was less cigarette smoking by those with Parkinson's disease.

One possible explanation for the observed inverse relationship is that PD may be under-reported as a cause of death in persons known to be smokers, simply because a suspected smoking-related cause of death would be more likely to be assigned. So, in mortality studies utilizing underlying cause of death, Parkinson's disease would be more likely to appear as a cause of death among non-smokers than smokers; spuriously resulting in an inverse relationship between the disease and smoking. Results from incidence studies (Godwin-Austen 1982), however, argue against this conjecture.

A clearcut beneficial effect of cigarette smoking is strongly supported by the scientific literature, although it is possible that this effect is spurious for reasons similar to those affecting studies demonstrating harmful effects. The effect appears in long-term incidence studies

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aimed at describing the detrimental effects of smoking, and in retrospective case-control studies directed specifically on investigating the protective effect of smoking noted previously in those prospective investigations. No obvious alternative explanation for these findings is supported by scientific evidence. Godwin-Austin, et al (1982) demonstrated the required temporal sequence, so it cannot be argued that the observed inverse relationship is due to PD patients selectively quitting smoking. No potential confounding factor which is associated with smoking and is predictive of PD has been identified. The results from prospective studies eliminate information bias due to underreporting of smoking by PD patients, and over-representation of smokers in control groups as reasons for the observed relationship and several possible biological mechanisms have been proposed. Additional research to identify the actual mechanism would be fruitful. The effect of smoking on reducing the incidence of Parkinson's disease is particularly tantalizing given the recent suggestions (Lewin 1987) that the disease is occurring at earlier ages. While this shift in age at onset might be due to increased exposures to some environmental hazard, one cannot easily dismiss the possibility that the recent reductions in the

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proportions of younger smokers might in part be responsible for these recent observations.

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OTHER DISEASES AND CONDITIONS

Although uncommon and accounting for about 1,300 deaths in the U.S. per year, cancer of the extrahepatic bile duct is another neoplastic disease which appears to be both hormonally related and less frequent in cigarette smokers. A recent epidemiologic case-control study (Yen 1987), which represents the best effort to-date to describe the etiology of the disease, comprises only 67 patients. Information on smoking, alcohol consumption, oral contraceptive use, and medical history was obtained by person interview of these patients and was compared with similar information obtained from 273 control patients with other ("non-tobacco-related") cancers. Compared to non-smokers,the rate ratio for current smokers was significantly reduced (RR=0.4, CI=0.18-0.86), and the rate ratio for ex-smokers was half that of non-smokers, though marginally non-significant. No differences in consumption of alcohol or other beverages between cases and controls was found. Histories of oral contraceptive use and of ulcerative colitis were statistically significantly associated with increased risk of extrahepatic bile duct cancer. The association with ulcerative colitis is interesting and

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consistent with the extensive evidence of a protective effect of smoking in the causation of the disease. One might expect the apparent protective effect of smoking on extrahepatic bile duct cancer to be stronger since the inverse relationship was especially prominent for "smoking-related" cancers, with a relative risk of 2.0 (1.3-3.0) for the lowest compared to highest quintile.

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NICOTINE AS A BEHAVIORAL AID

Warburton (1987) has argued elegantly for adoption of a functional view of smoking as a way to control one's psychological state; that is, as a benign form of self-medication rather than an irrational habit. He cites numerous studies of smoking motives and situations which support this functional model, and which indicate intrinsic constitutional differences between people who choose to smoke and those who do not. The functional view that smoking is the outcome of the interaction between a person and a situation derives from this evidence. The variation of one's smoking behavior in response to stressful events demonstrates the usefulness of smoking as a method of personal control of one's psychological state, and also of the efficiency of smoking as a nicotine delivery system providing rapid positive feedback. Studies of smoking cessation provide additional support for the functional view of smoking because they demonstrate that ex-smokers report an increase, after abstinence, in the effects they claim to avoid by smoking.

In several respects the effects of nicotine are similar in animals and humans, although the tissue distribution of nicotine differs among animal species and has not been determined in humans (Svensson 1987). In humans and animals, the drug has been found to have both

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a depressant and a stimulant action, depending on dosage and time after administration. Furthermore, based on animal experiments it appears that tolerance to the rewarding effects of nicotine does not develop (Iwamoto 1987). This, of course, most unexpected. Clark (1987) recently reviewed the evidence from animal studies which corroborates the theoretical and clinical evidence of the effects of nicotine on humans. He cites studies which have found decreased aggressive responding in humans and squirrel monkeys, depression of the consumption of sweet-tasting high-caloric foods in humans and rats, and improved mental performance and discrimination behavior in humans and rodents. Furthermore, he indicates that several of the behavioral actions of nicotine in animals are consistent with the effect of nicotine in alleviating anxiety and improving performance in humans.

Pharmacological experiments of the effects of nicotine on humans are of varied quality because of the methodological difficulties and ethical considerations inherent in this type of research. Individual variability and a complex relationship between habituation, perception, expectation, and personal control of dosage make interpretation of behavioral research on nicotine rather difficult. Nevertheless, the beneficial effects of nicotine on a variety of aspects of performance are well-documented in the scientific literature. Among the direct benefits are improved performance on certain

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tasks; skeletal muscle relaxation; stress, anxiety, and pain reduction; central nervous system arousal; appetite suppression and weight reduction, memory improvement; and mood elevation (Pomerleau 1986, Warburton 1986, Clark 1987, Domino 1987). These effects may be classified as improvements in 1) Vigilance, 2) Learning and memory, 3) Problem solving, 4) Motor control, and 5) Control of response to stressors. Although some of these effects clearly are inter-related, e.g. problem solving involves both attention and memory, this classification provides a useful framework for presenting the scientific literature on nicotine as a behavioral aid.

This presentation is not intended to be a comprehensive review, as several excellent reviews of the subject already are in the literature (Wesnes 1983). Rather, specific articles will be cited as examples of the evidence for or against a particular behavioral effect of nicotine. The reader should be aware of the numerous experimental parameters which affect the validity of this evidence. For example, the experimental approaches used in these investigations required the voluntary cooperation of a few subjects in a rigid controlled setting. Thus, the ability to generalize the studies' results may be limited, and one can question how well performance on tasks contrived for the experimental setting relates to the normal experience. Furthermore, timing is of critical importance in these studies because in general a pulse of relatively concentrated nicotine

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reaches the brain rapidly, it is rapidly diluted and has a very short biological half-life. In fact, there are so many places for significant variation in experimental design among studies that comparisons cannot easily separate real differences in their results from spurious ones due solely to different study designs.

Vigilance actually involves three distinct aspects, namely sustained attention, selective attention, and distraction avoidance. Testing of vigilance usually involves having a subject monitor a signal source for some unpredictable variation or specific sequence. The types of vigilance tests which have been employed vary widely, with the ideal being, as noted by Wesnes and Warburton (1983), a task in which one can demonstrate in a short testing period both impairments and improvements over time. Among the classic experiments are measurements based on the Continuous Clock Task (Macworth 1965) in which subjects are required to detect a brief pause in the passage of the second hand, and the "letter crossing test" (Wittenborn 1943) or its derivatives (Bakan 1959) which require picking out specific sequences. These tests measure sustained attention, and nicotine has been found to improve performance. In one experiment using the continuous clock method, heavy-, light-, and non-smokers were administered nicotine at 20, 40, and 60 minutes into an 80 minute session (Wesnes, et al 1983). This study compared the decline in sensitivity (vigilance) over time among the three groups, and found

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that nicotine reduced the decline significantly. Furthermore, the authors demonstrated that the effect was independent of smoking status. That is, smokers who abstain before a performance task do not appear to be dependent on nicotine to regain their previous level of function. Smokers were not merely returning to their baseline performance since the heavy smokers, the light smokers, and the non-smokers responded similarly to nicotine administration.

Wesnes and Warburton (1983) cite an early study of selective attention by Tarriere and Hartemann (1964), which combined "central guiding with peripheral visual surveillance" over a 2 1/2 hour time period. They point out that, although smokers who were allowed to smoke during the experiment missed far fewer peripheral signals than did smokers who did not smoke, it was not clear whether this was at the expense of reduced vigilance on the central guiding task. Nonsmokers' performance was intermediate between that of the two smoking groups, although their selective attention decreased significantly over time.

The classic example of experiments on this perceptual intrusion is the Stroop test, which determines the time difference between naming colors in congruous and incongruous situations. Evidence from studies of the effect of nicotine on reducing distraction during performance of vigilance tasks is consistent with that from other

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attention related studies (U.S.DHHS 1988). Knott (1985) investigated the role of tobacco as a "chemical stimulus filter" as reflected in electrocortical measures which are assumed to tap attentional processes. His study of female smokers revealed that tobacco-induced reductions of sensory evoked potentials differed by pre-experimental expired alveolar carbon monoxide (CO). The low CO group exhibited reduced potentials in the non-distracted experimental conditions, while the high CO group's reduction occurred only under the distracted condition.

The effects of nicotine on learning and memory, apart from its effect on attention, involve the acquisition, consolidation, and retention of information. Animal studies cited by Mangan (1983) suggest that nicotine facilitates learning and memory in all of these aspects, although the effect appears to be biphasic with smaller doses being facilitatory and larger ones inhibitory. Mangan (1983) found that there was an interaction between task difficulty and nicotine dosage resulting in impedance of learning an easy task and improvement at the same dose for learning a more difficult task. In contrast, he found that both low and medium doses enhanced recall in two types of learning tasks regardless of difficulty. Furthermore, data from Mangan and Golding (1983) suggest that the effect is to improve memory consolidation and is reflected in improvement of long-term, rather than short term memory.

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In two experiments of memory encoding and nicotine. Warburton, et al (1986) found that learning, i.e. input of information to storage, was facilitated by nicotine, and that it produced state-dependent learning. That is, behavior learned in one pharmacologic state is better remembered when retention is tested in the same state. Lowe's (1986) experiment with a combination of alcohol and nicotine ingestion confirmed this finding. Warburton, et al (1986) suggest that the reason why some studies have failed to find nicotine-related memory improvement is that these studies utilized different pharmacologic states for learning and testing. Results from Peeke and Peeke (1984), who found no effect of nicotine on memory, exemplify this methodological problem. More recently, Ney, et al (1988) have shown that experimental conditions other than pharmacologic state may affect the results of studies of cognitive performance and their applicability to real life situations because smokers modulate their intake to contend with task conditions. Thus, results from studies with experimental designs in which smoking is regulated or is not monitored may not reflect the effects of nicotine outside of the laboratory.

Problem solving ability involves learning, memory, and rapid information processing in which performance is measured by the speed and accuracy of responses. Wesnes and Warburton (1983)

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conducted two experiments to investigate the effects of of smoking on the performance of a rapid information processing task. The task involved detecting sequences of odd or even digits in a rapid series of psuedo-random digits. Their experiments on a small number of undergraduate volunteers demonstrated a greater improvement in performance after smoking a high nicotine cigarette than after lower nicotine cigarettes. In comparison with performance following a nicotine-free cigarette, nicotine-containing cigarettes improved performance. Furthermore, performance deteriorated over time among the subjects smoking nicotine-free cigarettes. Later experiments confirmed that higher yielding cigarettes produce greater improvements than lower yielding ones (Wesnes 1984b). These experiments were the first demonstration that smoking improves information processing performance above pre-smoking levels, rather than simply restoring it to those levels after abstinence. Other work by these authors (Wesnes 1984a , Wesnes & Revell 1984, Warburton 1984) supports the involvement of central cholinergic mechanisms in the processing of information in humans. Recent experimental evidence provides additional support to the involvement of the nicotinic cholinergic system in the processing of visual information or visual-motor function (London 1988).

Dunne, et al (1985) examined the effects of nicotine on problem solving performance among a group of non-smoking female

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volunteers. The subjects were required to solve both numerical and verbal problems of varying difficulties. This experiment demonstrated impairment of both recall and recognition of information learned subsequent to chewing nicotine gum. Nicotine had no influence on overall problem solving performance. Thus, the effects of nicotine on information processing may be cue and context specific rather than facilitating general mental efficiency. Alternatively, the negative results may have been due to the relative inefficiency of chewing gum as a nicotine delivery system or to the use of non-smokers as experimental subjects.

For over sixty years, smoking has been known to affect motor control by increasing hand tremor and by decreasing reaction time (U.S. DHHS 1988). Nicotine is the pharmocologic agent in tobacco smoke which is responsible for this, as demonstrated by West and Jarvis (1986), who administered nasal nicotine solution to non-smokers and found strong and consistent enhancement of motor control. Myrsten and Anderson (1978) examined the effect of smoking on tasks involving simple and complex reaction time. Their findings were similar to results from studies of problem solving performance, in that smoking prevented the deterioration of performance over time for the simple reaction time periods (i.e. prevented an increase in reaction time) and significantly improved (i.e. reduced) reaction time for complex tasks compared to the non-smoking condition. Morgan

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and Pickens (1982) investigated reaction time performance related to the smoking procedure used in various experiments. Although comparisons with non-smokers were not made, the authors found significantly slower reaction time associated with subjects' ad libitum smoking of their own cigarettes compared to ad libitum smoking or smoking with a prescribed puff pattern of standard cigarettes with higher nicotine content. While one might reasonably infer a nicotine dose-response relationship from this experiment, the importance of this work is in cautioning that experimental conditions may greatly affect the results of studies relating cigarette smoking to cognitive, behavioral, and physical function.

Well documented and perhaps one of the most important positive effects of nicotine is its apparent usefulness in an individual's ability to control response to stressors (Gilbert 1979). This is accomplished through reduction of negative feelings or emotions (or in psychological terms, negative affect) and enhancement of positive ones. However, Lombardo and Epstein (1986) have pointed out that because nicotine also increases sympathetic nervous system activity, empirical research on the physiology of emotion suggests that nicotine should increase negative affect. They tested one hypothesized explanation for this paradox, i.e. interference by nicotine with the perception of autonomic arousal, and failed to find support. It is likely that a smoker's perception or expectation of the

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relaxing effect of cigarette smoking determines its actual effect to a some degree. If this is the result of a conditioned response, then a study's failure to find such an effect (Hatch 1983) may be due to the parameters of an experimental setting.

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